



The Health Case for Reforming the Toxic Substances Control Act

By reforming the Toxic Substances Control Act of 1976, we have the potential to reduce Americans' exposure to toxic chemicals, improve health, and lower health care costs.

Acknowledgments

This report is the result of a collaborative effort by members of the Safer Chemicals, Healthy Families coalition, a new nationwide effort to adopt smart federal policies that protect us from toxic chemicals. (www.saferchemicals.org).

The Health Case for Reforming the Toxic Substances Control Act relies primarily on five peer-reviewed studies and reviews of the scientific literature that collectively reference more than 1,200 published papers and reports. We thank the authors of these papers for their seminal work:

R.W. Clapp, M.M. Jacobs, and E.L. Loechler “Environmental and Occupational Causes Of Cancer: New Evidence 2005–2007,” *Reviews on Environmental Health*, 23, no.1 (2008): 1–47.

Jill Stein, et al., *Environmental Threats to Healthy Aging: With a Closer Look At Alzheimer’s and Parkinson’s Disease*, ed. Nancy Myers (Boston: Greater Boston Physicians for Social Responsibility and the Science and Environmental Health Network, 2008).

Philippe Grandjean and Philip J. Landrigan, “Developmental Neurotoxicity of Industrial Chemicals,” *Lancet*, 368, no. 9553 (December 2006): 2167–78.

D. Andrew Crain, et al., “Female Reproductive Disorders: the Roles of Endocrine-Disrupting Compounds and Developmental Timing,” *Fertility and Sterility*, 90, no. 4 (2008): 911–40.

Mark J. Mendell, “Indoor Residential Chemical Emissions as Risk Factors for Respiratory and Allergic Effects in Children: A Review,” *Indoor Air*, 17, no. 4 (2007): 259–77.

NOTE: When this report includes a statement that is not based on one of these review articles, the statement is separately cited.

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- Caroline Baier-Anderson, PhD, Environmental Defense Fund
- Charlotte Brody, RN, Safer Chemicals, Healthy Families Coalition
- Richard Denison, PhD, Environmental Defense Fund
- Susanne Frank, Safer Chemicals, Healthy Families Coalition
- Ruth Hennig, John Merck Fund
- Elizabeth Hitchcock, U.S. Public Interest Research Group
- Molly Jacobs, MPH, Lowell Center for Sustainable Production, University of Massachusetts Lowell
- Christine James, MA, John Merck Fund
- Sarah Janssen, MD, PhD, MPH, Natural Resources Defense Council
- Margie Kelly, Safer States Coalition, Safer Chemicals, Healthy Families Coalition
- Joyce Martin, JD, American Association on Intellectual and Developmental Disabilities
- Elise Miller, MEd, Collaborative on Health and the Environment
- Daniel Rosenburg, JD, Natural Resources Defense Council
- Kathleen Schuler, MPH, Institute for Agriculture and Trade Policy
- Gina Solomon, MD, MPH, Natural Resources Defense Council
- Maureen Swanson, MPA, Learning Disabilities Association of America
- Tracey Woodruff, PhD, MPH, Program on Reproductive Health and Environment, University of California, San Francisco

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Executive Summary

There is growing agreement across the political spectrum that the Toxic Substances Control Act (TSCA) of 1976 does not adequately protect Americans from toxic chemicals. In the 34 years since TSCA was enacted, the EPA has been able to require testing on just 200 of the more than 80,000 chemicals produced and used in the U.S., and just five chemicals have been regulated under this law. Environmental Protection Agency (EPA) Administrator Lisa Jackson has asked Congress to provide her agency with better chemical management tools for safeguarding our nation's health.¹

Much has changed since TSCA became law more than 30 years ago. Scientists have developed a more refined understanding of how some chemicals can cause and contribute to serious illness, including cancer, reproductive and developmental disorders, neurologic diseases, and asthma.

The Safer Chemicals, Healthy Families coalition believes that, by reforming TSCA, we can reduce our exposure to toxic chemicals, improve our nation's health, and lower the cost of health care. This report documents some of the scientific findings and economic analysis underlying our position.

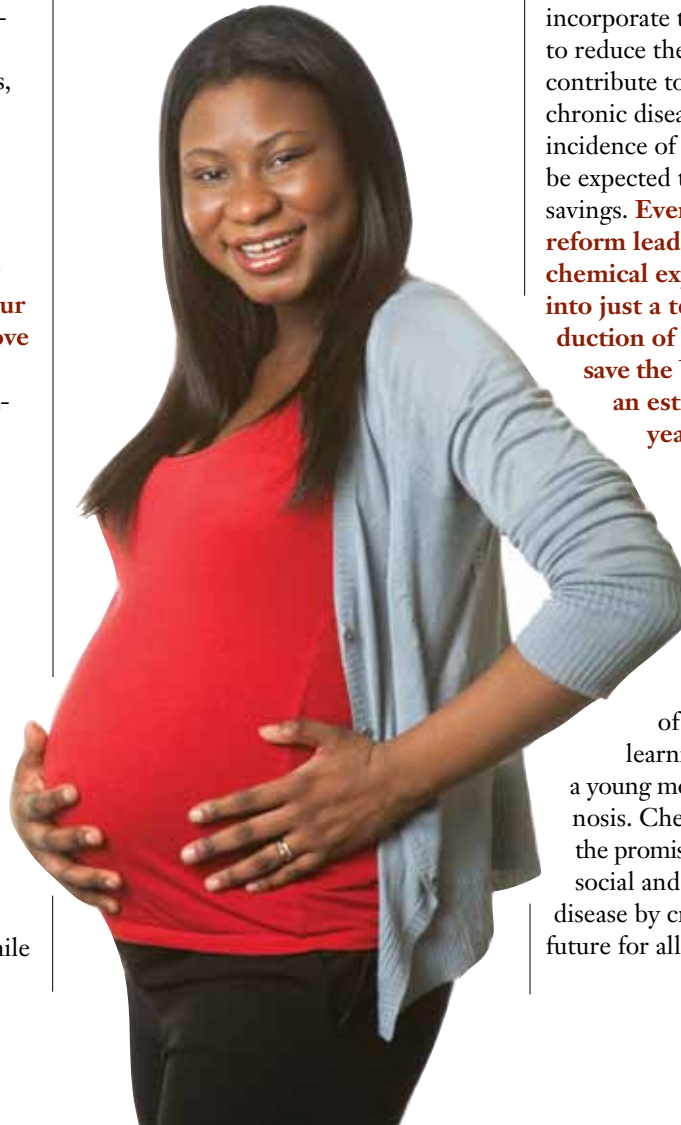
Chronic disease is on the rise

More than 30 years of environmental health studies have led to a growing consensus that chemicals are playing a role in the incidence and prevalence of many diseases and disorders in our country, including:

- **Leukemia**, brain cancer, and other childhood cancers, which have increased by more than 20% since 1975.²
- **Breast cancer**, which went up by 40% between 1973 and 1998.³ While breast cancer rates have declined

since 2003, a woman's lifetime risk of breast cancer is now one in eight, up from one in ten in 1973.⁴

- **Asthma**, which approximately doubled in prevalence between 1980 and 1995 and has stayed at the elevated rate.^{5,6}
- **Difficulty in conceiving and maintaining a pregnancy** affected 40% more women in 2002 than in 1982. The incidence of reported difficulty has almost doubled in younger women, ages 18–25.^{7,8}
- **The birth defect resulting in undescended testes**, which has increased 200% between 1970 and 1993.⁹
- **Autism**, the diagnosis of which has increased more than 10 times in the last 15 years.¹⁰



The health and economic benefits of reforming TSCA

According to the U.S. Centers for Disease Control and Prevention (CDC), 133 million people in the U.S.—almost half of all Americans—are now living with these and other chronic diseases and conditions, which now account for 70% of deaths and 75% of U.S. health care costs.¹¹

Estimates of the proportion of the disease burden that can be attributed to chemicals vary widely, ranging from 1% of all disease¹² to 5% of childhood cancer¹³ to 10% of diabetes, Parkinson's disease, and neurodevelopmental deficits¹⁴ to 30% of childhood asthma.¹⁵ Whatever the actual contribution, effective chemical policy reform will incorporate the last 30 years of science to reduce the chemical exposures that contribute to the rising incidence of chronic disease. And any decline in the incidence of chronic diseases can also be expected to bring health care cost savings. **Even if chemical policy reform leads to reductions in toxic chemical exposures that translate into just a tenth of one percent reduction of health care costs, it would save the U.S. health care system an estimated \$5 billion every year.**

The U.S. now spends over \$7,000 per person per year directly on health care.¹⁶ This sum does not include the many other kinds of costs, such as the costs of raising a child with a severe learning disability or coping with a young mother's breast cancer diagnosis. Chemical policy reform holds the promise of reducing the economic, social and personal costs of chronic disease by creating a more healthy future for all Americans.

Introduction



There is a growing consensus among scientists, health care providers, health and environment advocates, consumer product companies, and even the chemical industry that, when it comes to protecting Americans from toxic chemicals, current law has not kept up with the times.

The primary chemical safety law, the Toxic Substances Control Act of 1976 (TSCA), gave all chemicals that were in existence prior to 1976 a free pass by not requiring any testing for safety in order to remain on the market. In the 34 years since TSCA was enacted, the Environmental Protection Agency has required testing on just 200 of the more than 80,000 chemicals produced and used in the U.S. over that same period.

Much has changed since 1976: chemicals have become more pervasive in daily life and scientists have developed a more complex understanding of how people are exposed to chemicals and how such exposures can contribute to serious illness, including cancer, reproductive and developmental disorders, neurological diseases, and asthma.

Making the health care case for reform

The members of the Safer Chemicals, Healthy Families coalition believe that, by reforming TSCA, we can reduce exposure to toxic chemicals, improve our nation's health, and lower health care costs. This report summarizes some of the scientific studies documenting that chemicals are contributing to the growing burden of chronic disease in our

country, and offers an analysis of the economic benefits of reform.

Specifically, we review the growing scientific literature linking chemical exposures to six categories of chronic conditions that impact the daily lives of millions of Americans: certain types of cancer, learning and developmental disabilities, Alzheimer's and Parkinson's disease, reproductive health and fertility problems, and asthma. This report also incorporates the results of published studies that estimate the portion of our disease burden that is attributable to chemical exposures and the potential health cost savings from improved protection from toxic chemicals.

THE COST TO OUR HEALTH

Cancer

Cancer affects millions of American families and adds billions of dollars to our nation's annual health care bill. According to current statistics from the National Cancer Institute (NCI), over 11 million people are living with cancer in the U.S.¹⁷ NCI estimates that 44% of men and 38% of women in the U.S. will be diagnosed with cancer at some point in their lives.¹⁸

Cancer is the second most common cause of death in the U.S., exceeded only by heart disease. More than 1.5 million people were diagnosed with new cases of cancer in 2009. In 2008 the direct medical costs of cancer were \$93.2 billion and the overall costs were \$228.1 billion.^{19,20} Medical costs for pediatric cancers alone in 1997 totaled an estimated \$3.9 billion.²¹

Over the past two decades, the rates of some cancers rose significantly. These include:

- Kidney, liver, thyroid, esophageal and testicular cancer, as well as melanoma in men.
- Non-Hodgkin's lymphoma, Hodgkin's disease, melanoma and cancers of the thyroid, liver, and kidney in women.
- Childhood cancers overall, especially childhood leukemia and brain cancer (see Figure 1).

Cancer and chemicals in the workplace

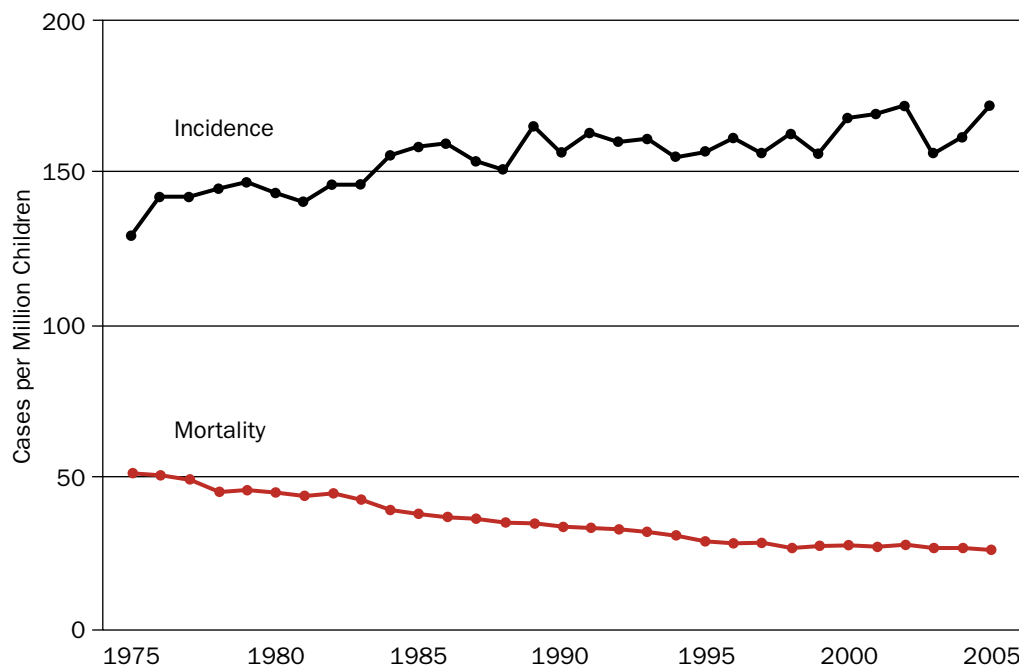
Much of what we know about chemicals and cancer comes from studies of workers who were diagnosed with cancer after exposure to chemicals in their place of employment. The U.S. Department of Health and Human Services Report on Carcinogens lists chemical hazards

such as asbestos, hexavalent chromium, and vinyl chloride as known human carcinogens, and formaldehyde, trichloroethylene (TCE), and methylene chloride (dichloromethane) as likely human carcinogens.²² The classifications of these and the other chemicals listed in Table 1 are largely based on studies of similarly exposed and diagnosed workers. These human studies both confirm and are confirmed by animal testing and other research findings on the same chemicals.

Looking beyond workplace exposure

In the three decades since TSCA became law, it has become clear that the American people's exposure to these and other cancer-causing chemicals is not limited to occupational settings. In an Arizona study designed to be representative of the general U.S. population,

FIGURE 1 **Cancer Incidence and Mortality for Children Under 20**



Cancer is the second most common cause of death for Americans under the age of 20. The incidence of childhood cancer increased more than 20% between 1975 and 1990. Since 1990, the incidence has remained roughly at this elevated rate. Although improved treatment options have led to a decline in the percentage of children who die from cancer, the percentage of children who are diagnosed with leukemia, brain, and other cancers has increased.

Source: U.S. EPA. America's Children and the Environment. www.epa.gov/envirohealth/children
Data: National Cancer Institute, Surveillance, Epidemiology and End Results Program

researchers found that 25% of homes contained formaldehyde levels above recommended exposure levels.²⁴ Formaldehyde, a known human carcinogen encountered in the workplace, is also a common indoor air contaminant because of its use in furniture, cabinets, countertops, insulation, wallpaper, paints, and paneling. Formaldehyde is found in a wide variety of consumer products such as antiseptics, medicines, cosmetics, nail polish, dishwashing liquids, fabrics and fabric softeners, shoe-care agents, carpet cleaners, glues and adhesives, lacquers, paper, coatings, and plastics.²⁵

Trichloroethylene (TCE) is another widely used chemical that is a likely human carcinogen. It is found in occupational settings as well as in consumer products such as paints and paint removers, adhesives, rug cleaners, typewriter correction fluids, metal cleaners, pepper sprays, and spot removers.²⁶

TCE is one of the most common contaminants at toxic waste sites and is a common contaminant of groundwater aquifers. According to the Agency for Toxic Substances and Disease Registry (ATSDR), between 9% and 34% of drinking water supply sources tested in the U.S. contain some TCE.²⁷ In one study, TCE was found in all samples of human breast milk from urban areas in the U.S.²⁸

In the three decades since TSCA became law, it has become clear that the American people's exposure to these and other cancer-causing chemicals is not limited to occupational settings.



TABLE 1 Commonly Found Chemicals Known or Reasonably Anticipated to Be Human Carcinogens²³

- Arsenic
- Asbestos
- Benzene
- Benzidine
- Butadiene
- Cadmium
- Carbon Tetrachloride
- Chromium (hexavalent)
- Coal Tars
- Ethylene oxide
- Formaldehyde
- Lead
- Methylene Chloride
- Nickel
- Silica
- Styrene-7,8-oxide
- Sulfuric Acid
- Toluene Diisocyanate
- Trichloroethylene (TCE)
- Vinyl Chloride

Early exposure and later cancer

Recent research has revealed how early life exposures can lead to cancer many decades later, a finding that suggests that not all carcinogens can be identified through worker exposures. Among the new studies that show the importance of the timing of exposure is a 2007 report that found a linkage between early exposure to DDT (dichloro diphenyl trichloroethane, a pesticide) and later development of breast cancer.²⁹

Researchers have been investigating the relationship between DDT and breast cancer for decades.³⁰ Older studies looked at the relationship between breast cancer and the levels of DDT in women at the time of breast cancer diagnosis. Those studies did not find a strong connection between DDT and breast cancer. But in 2007, researchers published a new study of women with breast cancer that documented the DDT levels these women were exposed to when they were younger than 14 years old, through analysis of stored

blood samples. The study vividly illustrates that the timing of exposure matters. Women who were exposed to DDT at ages younger than 14 had an increased risk of breast cancer. But for women exposed only after the age of 14, the increased risk from DDT was essentially zero. This study demonstrates that when children or adolescents are exposed during vulnerable periods of development, chemicals can have profound effects that are not manifest until later in life.³¹

Breast cancer rates in the U.S. increased by more than 40% between 1973 and 1998, and though in the last several years there has been a slight decline in breast cancer incidence, it remains one of the leading causes of death in women. Today, a woman's lifetime risk of breast cancer is one in eight, up from 1 in 10 in 1973.³² The human study described above has been modeled in laboratory animals, where early life exposures to low doses of chemicals have been shown to increase the risk for breast cancer by affecting mammary development and lifetime susceptibility to cancer. In laboratory studies, bisphenol A is one of the chemicals that has also been shown to cause normal breast tissue to express genes associated with a highly aggressive, and often fatal, form of breast cancer.³³

Reforming TSCA to close gaps in knowledge

While there has been a marked increase in the number of published studies on the connection between chemical exposure and cancer, researchers continue to be thwarted by all that is *not* known about the many chemicals that pervade daily life. Under TSCA, EPA has only required testing on 200 of the 80,000 chemicals that have been produced and used in the U.S. Most of the chemicals in use today were simply grandfathered in when TSCA became law in 1976, with no requirement that they be tested or shown to be safe. So we do not know how many other chemicals may act like

DDT, leading to cancer many decades after exposure.

To be effective, TSCA reform must require chemical manufacturers to provide information on the health hazards associated with their chemicals, how they are used, and how much the public or workers could be exposed. Chemical manufacturers need to be made responsible for demonstrating the safety of their products.

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TSCA's failure:

The asbestos example

Effective TSCA reform will give EPA the power to restrict dangerous substances like asbestos, a silicate material that has been banned in 40 other countries.³⁴ In 1989, after a ten-year, \$10 million cost-benefit analysis and the development of a 100,000 page administrative record, EPA attempted to ban asbestos. But a federal court in 1991 overturned the ban, ruling that TSCA requires that EPA not only show that a chemical is harming human health but also that the proposed restriction is the "least burdensome alternative" for eliminating "an unreasonable risk." Since that ruling, EPA has never tried again to ban a substance using its TSCA authority.³⁵ To protect public health, TSCA reform should give EPA the power to restrict asbestos and other known human carcinogens to which people are exposed. New legislation should require EPA to assess chemicals against a health-based standard that is designed to protect the health of even the most vulnerable Americans.³⁶



THE COST TO OUR HEALTH

Learning and Developmental Disabilities

Learning and developmental disabilities (LDDs) are estimated to affect approximately one in six children under age 18 in the U.S. These neurodevelopmental disorders appear to be rising, but more data is needed to confirm that conclusion. Intellectual disability (ID, formerly referred to as mental retardation) impacts 2%, or approximately 1.4 million, children. Attention deficit hyperactivity disorder (ADHD) is conservatively estimated to occur in 3–6%, or approximately 2 million, children. Almost 1% of 8-year-old children are diagnosed with autism spectrum disorder, a 10-fold increase over just a 15-year period.^{37,38,39} About 30% of this dramatic rise in autism cannot be explained by changes in the age of diagnosis and the inclusion of milder cases.⁴⁰

These conditions impose tremendous psychological and economic costs on the affected children, their families, and communities. Just the cost of providing special education services to students with disabilities amounted to \$77.3 billion in 1999–2000, an average of \$12,474 per student.⁴¹ According to the U.S. Centers for Disease Control and Prevention (CDC) individuals with an autism spectrum disorder have average medical expenditures that exceed those without the disorder by \$4,110–\$6,200 per year.⁴² A 2006 study reported that the economic costs associated with autism in the U.S. are approximately \$35 billion dollars per year.⁴³

Chemicals, learning and developmental disabilities, and TSCA

Research on occupational exposures and epidemics of industrial chemical poisoning have led to the identification of lead, methyl-mercury, polychlorinated biphenyls (PCBs), arsenic, and toluene as known causes of neurodevelopmental disorders. Industrial chemicals have been identified in the peer reviewed

TABLE 2 Chemicals Known to be Neurotoxic to Humans

Metals and inorganic compounds	Organic solvents	Other organic substances
<ul style="list-style-type: none"> Aluminum compounds Arsenic and arsenic compounds Azide compounds Barium compounds Bismuth compounds Carbon monoxide Cyanide compounds Decaborane Diborane Ethylmercury Fluoride compounds Hydrogen sulphide Lead and lead compounds Lithium compounds Manganese and manganese compounds Mercury and mercuric compounds Methylmercury Nickel carbonyl Pentaborane Phosphine Phosphorus Selenium compounds Tellurium compounds Thallium compounds Tin compounds 	<ul style="list-style-type: none"> Acetone Benzene Benzyl alcohol Carbon disulphide Chloroform Chloroprene Cumene Cyclohexane Cyclohexanol Cyclohexanone Dibromochloropropane Dichloroacetic acid 1,3-Dichloropropene Diethylene glycol N,N-Dimethylformamide 2-Ethoxyethyl acetate Ethyl acetate Ethylene dibromide Ethylene glycol n-Hexane Isobutyronitrile Isophorone Isopropyl alcohol Isopropylacetone Methanol Methyl butyl ketone Methyl cellosolve Methyl ethyl ketone Methylcyclopentane Methylene chloride Nitrobenzene 2-Nitropropane 1-Pentanol Propyl bromide Pyridine Styrene Tetrachloroethane Tetrachloroethylene Toluene 1,1,1-Trichloroethane Trichloroethylene Vinyl chloride Xylene 	<ul style="list-style-type: none"> Acetone cyanohydrin Acrylamide Acrylonitrile Allyl chloride Aniline 1,2-Benzenedicarbonitrile Benzonitrile Butylated triphenyl phosphate Caprolactam Cyclonite Dibutyl phthalate 3-(Dimethylamino)-propanenitrile Diethylene glycol diacrylate Dimethyl sulphate Dimethylhydrazine Dinitrobenzene Dinitrotoluene Ethylbis(2-chloroethyl)amine Ethylene Ethylene oxide Fluoroacetamide Fluoroacetic acid Hexachlorophene Hydrazine Hydroquinone Methyl chloride Methyl formate Methyl iodide Methyl methacrylate p-Nitroaniline Phenol p-Phenylenediamine Phenylhydrazine Polybrominated biphenyls Polybrominated diphenyl ethers Polychlorinated biphenyls Propylene oxide TCDD Tributyl phosphate 2,2',2"-Trichlorotriethylamine Trimethyl phosphate Tri-o-tolyl phosphate Triphenyl phosphate

scientific literature as causing neurological effects in humans, mostly through occupational exposures (see Table 2). Many of these chemicals are in common use and are produced in high volumes.

The human brain: more susceptible during development

In the years since TSCA became law, evidence has been accumulating that lead, mercury and other neurotoxic chemicals have a profound effect on the developing brain at levels that were once thought to be safe. Scientists have learned that the developing human brain is much more susceptible to toxic substances than the adult brain. Windows of unique vulnerability occur as the brain begins to develop *in utero* and continue through adolescence, along a precise and delicate step-by-step sequence involving various neurobiological processes. A chemical exposure at three months gestation may result in

a different effect than exposure to the same chemical at six months gestation or at two years of age. If chemicals inhibit, interfere with, or halt a developmental process, the damage may be permanent. This new science suggests that industrial chemicals could be creating a pandemic of subclinical neurotoxicity—harm to the brain and nervous system that is not linked to a specific clinical diagnosis.

This new understanding of subclinical neurotoxicity also suggests that there may be a thousand or more other chemicals that can impact the developing brain, although no authoritative estimate of the true number of neurotoxicants is available. Since TSCA was enacted, very little data has been collected on the effects of chemicals on the developing nervous system. For most of the 3,000 chemicals produced in highest volume (over one million

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pounds per year), only 12 have been adequately tested for neurotoxicity. To ensure healthy brain development for future generations, TSCA must be updated to require that all existing and new chemicals are tested and shown to be safe for pregnant women, children, workers, and other vulnerable populations.⁴⁴



THE COST TO OUR HEALTH

Alzheimer's and Parkinson's Disease

Alzheimer's disease is estimated to affect nearly 4.5 million people in the U.S. About 5% of men and women aged 65–74 have Alzheimer's disease, while nearly half of those aged 85 and older may have the disease. By 2050, researchers estimate that this number will nearly triple to over 13 million.

About 50,000 new cases of Parkinson's disease are reported annually in the U.S. Lack of patient registries, however, makes it difficult to estimate incidence and trends of the disease. The range of reported incidence of Parkinson's disease varies from 4.5 to 21 per 100,000 people annually. The prevalence of Parkinson's is expected to double by 2030.

The Alzheimer's Association estimates that national direct and indirect annual costs of caring for individuals with Alzheimer's disease are nearly \$150 billion. The cost of Parkinson's disease in the U.S. is estimated to be \$13–28.5 billion per year.

The link to chemical exposure

As described previously, the scientific literature identifies more than 100 industrial chemicals that are known to affect the human brain and nervous system, causing memory, cognitive, and functional symptoms.⁴⁵ While most of these studies have not focused on the aging brain, recent findings on lead, aluminum, PCBs, pesticides, particulate air pollution, and solvents suggest that chemicals affecting the developing brain may be harming the aging brain as well, leading to cognitive disorders like Alzheimer's disease and Parkinson's-like symptoms.

For example, in one recent study, 21% of more than a thousand patients presenting to a university clinic for cognitive disorders had medical histories that suggested they may have been exposed



to chemicals in their workplace or from an environmental source. Clinicians found that a history of toxic exposure was associated with cognitive decline at significantly younger ages.⁴⁶

Another study divided a population of elderly men into four groups, based on the amount of lead found in the bones of their kneecaps. Researchers found that each increasing amount of bone lead was associated with accelerated cognitive aging; the most exposed group had 15 years of additional cognitive aging compared to the lowest exposed group. This study suggests that lead has a substantial impact on cognitive aging across the population.⁴⁷

A series of animal studies on lead suggest that early-life lead exposure may contribute to late-life neurodegeneration. Rodents exposed to lead prenatally exhibited increases in Alzheimer-asso-

ciated abnormal brain proteins later in life. In contrast, exposure to lead during old age did not cause increases in the Alzheimer's disease-related proteins. The same delayed, late-life increase in Alzheimer's disease-related proteins was reported in aged monkeys exposed in infancy to low levels of lead.^{48,49}

New animal studies have also resurrected the 1960s controversy about the role of aluminum in neurodegenerative disease. One small study showed that when rodents were chronically exposed to dietary aluminum (similar to typical human exposure levels), aluminum accumulated in the brain. A larger follow-up study in rats showed that the more aluminum a rat received in its diet, the more memory loss the rat exhibited.^{50,51}

PCBs and the developing brain

Polychlorinated biphenyls (PCBs), may also be playing a role in the incidence of

both Alzheimer's and Parkinson's disease. PCBs are a group of chemicals that have been shown to harm the developing brain. PCBs were used for many years as paint additives, lubricants, and insulators in electrical equipment before they were banned in 1976. Only three published epidemiologic studies have explored the effects of PCBs on cognitive decline or dementia in older human subjects. Each of these studies found an association of adult PCB exposure with dementia or cognitive impairment.

PCBs have also been tied to Parkinson's disease. A retrospective mortality study of over 17,000 workers occupationally exposed to PCBs reported a nearly three-fold greater incidence of Parkinson's disease-related deaths than expected. Twice as many dementia-related deaths were seen in the women most highly exposed to PCBs—but not in men, even though men are generally at higher risk of Parkinson's disease. Another post-mortem study found higher levels of PCBs in the brains of people with Parkinson's disease than in controls.⁵² Animal and cellular studies have also shown that some PCBs produce Parkinson-like changes in the brain or brain cells.

Solvents and Parkinson's disease

A variety of solvents are used for cleaning, degreasing, extraction, surface coating, and laboratory work. Solvents are also components of paints, inks, glues, adhesives, and hydrocarbon fuels. Several solvents, including carbon disulfide, methanol, n-hexane, and trichloroethylene (TCE), have been reported to be associated with Parkinson's disease. One recent report describes Parkinsonism in 30 workers associated with long-term occupational exposure to TCE.⁵³ TCE is a particular concern because of widespread exposure: it is frequently used as a degreasing agent in industry, and is also a common surface- and ground-water contaminant, resulting in widespread, low-level exposures in the general population.

TSCA, Alzheimer's and Parkinson's

When TSCA was enacted in 1976, it banned new production and use of PCBs because of the accumulating evidence indicating they could cause cancer. Now we are learning that PCBs are among the chemicals that may be contributing to neurological disease as well.

More than 30 years after the ban, PCBs continue to contaminate the environment because they are persistent and not easily broken down. And because they are fat-soluble and bioaccumulative, they also continue to enter and contaminate the general food supply, which serves as an ongoing source of human exposure.

Biomonitoring data from the Centers for Disease Control and Prevention (CDC) show that the American public is still widely contaminated with PCBs. While the levels of PCBs in the general population are going down, hotspots of PCB contamination still exist across the U.S.⁵⁴

To be effective, TSCA reform must recognize the unique dangers posed by exposure to persistent, bioaccumulative toxins (PBTs), like PCBs, and include provisions to phase out all non-essential uses of them. Communities and populations that bear disproportionately high burdens of PBT contamination need to be the focus of exposure reduction efforts.⁵⁵



THE COST TO OUR HEALTH

Reproductive Health and Fertility Problems

In the U.S. today, there is increasing concern that environmental contaminants may be harming the reproductive health and fertility of women and men. Reproductive and fertility problems appear to be on the rise:

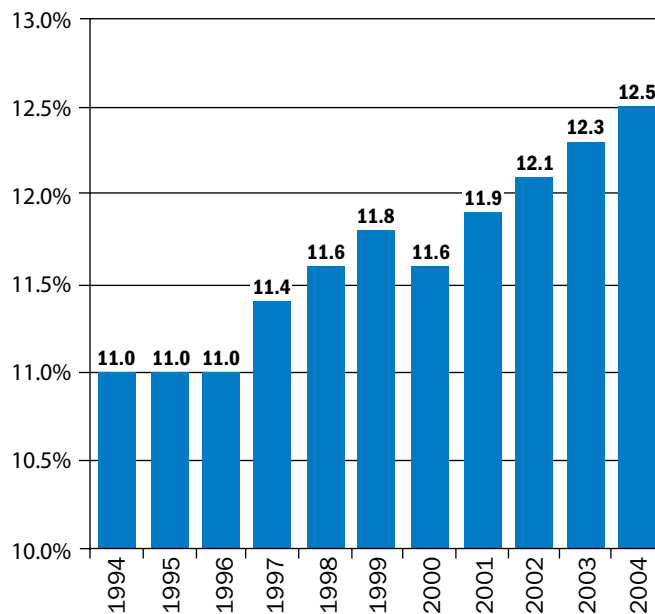
In women:

- At least 12% of women reported difficulty in conceiving and maintaining pregnancy in 2002, an increase of 40% from 1982. The rate has almost doubled in younger women, ages 18–25.⁵⁶
- Uterine fibroids are the number one cause of hysterectomy in reproductive-aged women, accounting for more than 200,000 of these surgeries annually in the U.S. alone. In addition, uterine fibroids are a significant cause of pelvic pain, heavy menstrual bleeding, abnormal uterine bleeding, infertility, and pregnancy complications.
- Fibroids and other fertility-related diseases, like endometriosis and polycystic ovarian syndrome, are diagnosed more frequently now, which may result from a true increase, better detection, or both.

In men:

- According to a large study of men from the Boston area, testosterone levels in adult men are declining. This decline is not explained by an increase in age or other health or lifestyle factors such as obesity or smoking.⁵⁷
- Testicular cancer increased by 60% between 1973 and 2003 in the U.S.⁵⁸
- There have been significant declines in sperm counts in men in the U.S., Europe, and Australia.⁵⁹

FIGURE 2 Percent of Preterm Births by Year, 1994–2004



Source: T.J. Mathews and Marian F. MacDorman, *Infant Mortality Statistics from the 2003 Period Linked Birth/Infant Death Data Set*, National Vital Statistics Reports 54 (16) (May 2006) Robert L. Goldenberg and Dwight J. Rouse, “Prevention of Premature Birth,” *New England Journal of Medicine* 339 (5) (1998): 313–20; Bernard Weiss and David C. Bellinger, “Social Ecology of Children’s Vulnerability to Environmental Pollutants,” *Environmental Health Perspectives* 114 (10) (2006): 1449–85.

Taken from the report, *Reproductive Roulette*, published by the Center for American Progress, 2009.

In children:

- Reproductive tract abnormalities are increasing in certain populations. Cryptorchidism (undescended testicles) increased 200% between 1970 and 1993.⁶⁰
- On average, babies are now born one week earlier than they were 15 years ago. And 30% more babies are born prematurely—the major reason more babies are being born with low birth weight.⁶¹ (See Figure 2).
- There is evidence of a trend in the U.S. toward earlier breast development and onset of menstruation in girls. A weight-of-the-evidence evaluation of human and animal studies suggests that endocrine-disrupting chemicals, particularly estrogen mimics and antiandrogens, as well as increased body fat, are important factors associated with altered puberty timing.⁶²

Health care costs

In 2002, U.S. patients and their insurers spent an estimated \$2.9 billion on infertility treatments.⁶³ Hysterectomies for uterine fibroids cost Americans \$1.7 billion per year.

In 2005, preterm birth cost the U.S. at least \$26.2 billion, or \$51,600 for every infant born prematurely. The costs broke down as follows:

- \$16.9 billion (65%) for medical care
- \$1.9 billion (7%) for maternal delivery
- \$611 million (2%) for early intervention services
- \$1.1 billion (4%) for special education services
- \$5.7 billion (22%) for lost household and labor market productivity⁶⁴

The average first-year medical costs, including both inpatient and outpatient care, were about 10 times greater for preterm infants (\$32,325) than for full-term infants (\$3,325).

The link to chemical exposure

The Centers for Disease Control and Prevention (CDC) has published data showing that exposures to chemicals like phthalates, bisphenol A (BPA), perfluorinated compounds, and cadmium are common. The CDC reports

Over the past decade, a wealth of new studies has shown how some chemicals can act as endocrine disruptors—chemicals that interfere with normal hormone function and regulation.

that almost everyone has these chemicals in their bodies—some at levels near or above those shown in scientific studies to cause adverse effects on reproductive health.

Over the past decade, a wealth of new studies has shown how some chemicals can act as endocrine disruptors—chemicals that interfere with normal hormone function and regulation. Among these are animal studies that link prenatal and early-life exposures to BPA found in polycarbonate plastic and food can linings to permanent reproductive changes and increased risks of later reproductive health problems, such as infertility, early puberty, breast cancer, and prostate cancer.

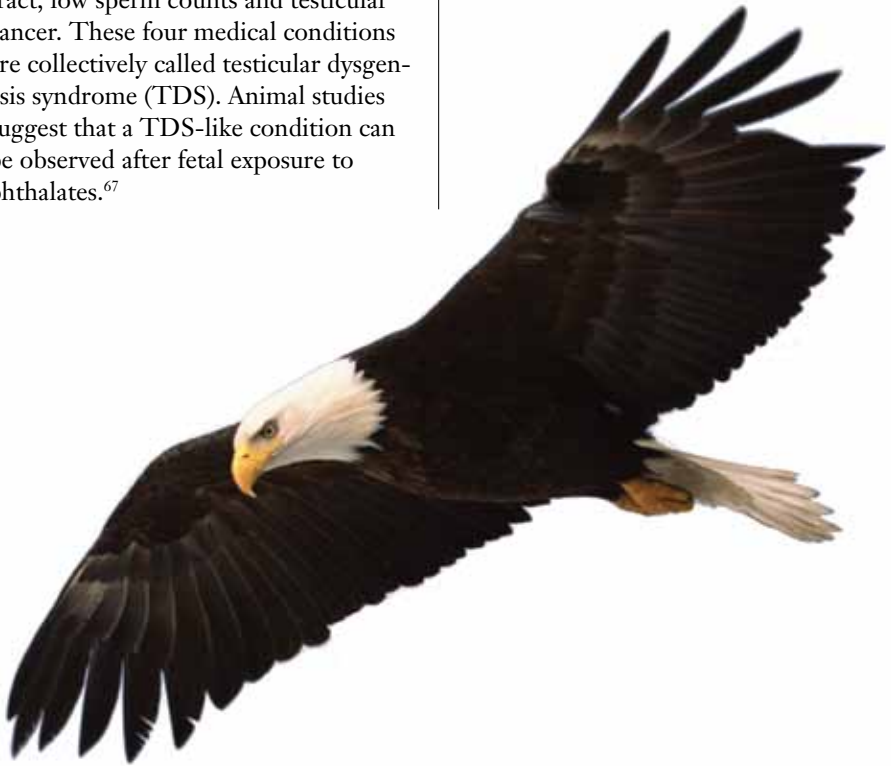
In laboratory animals, including non-human primates, BPA has previously been identified to affect development of the brain, causing changes in gender specific behaviors. Recently, a new human study reported that the higher a pregnant woman's BPA levels were during her first 16 weeks of pregnancy,

the more likely her child was to later show behavior somewhat atypical of its gender at age two. Girls engaged in more masculinized behaviors, while boys were more feminized.⁶⁵ Another new study of Chinese men exposed to higher levels of BPA in their workplace suggests that BPA can harm the reproductive health of adults as well. The report found substantially more sexual dysfunction among the exposed workers than a control group, including four times more erectile dysfunction and seven times more difficulty ejaculating.⁶⁶

Prenatal exposure to phthalates found in personal care products and in items made from vinyl has been linked to birth defects of the male reproductive system and feminized behaviors in boys. Some researchers now group the male birth defects—undescended testes (cryptorchidism) and deformities of the penis (hypospadias)—with two other conditions of the male reproductive tract, low sperm counts and testicular cancer. These four medical conditions are collectively called testicular dysgenesis syndrome (TDS). Animal studies suggest that a TDS-like condition can be observed after fetal exposure to phthalates.⁶⁷

In November 2009, a new study of mothers and their children found that boys born to mothers with higher levels of phthalates in their urine during pregnancy were more likely to exhibit feminized behaviors than boys whose mothers had lower levels of exposure.⁶⁸ Two other studies have also suggested that phthalate levels in young girls are linked to early breast development.^{69,70}

Low levels of prenatal exposure to perfluorinated chemicals, commonly used in stain-proof and stick-free products, were tied to low birth weight and body mass in newborns born at a city hospital in Baltimore. Babies with higher levels of these compounds tended to be slightly but significantly smaller than those with lower exposure.⁷¹ A recent study found that Danish men with higher levels of perfluorinated compounds had fewer normal sperm and lower sperm concentrations.⁷²



A wide range of wildlife populations has been adversely affected by exposure to endocrine-disrupting contaminants. Impacts among birds, fish, shellfish, mammals, and reptiles include decreased fertility and increased reproductive tract abnormalities; feminization and demasculinization in the males; and masculinization and defeminization in the females.⁷⁴

Cadmium, a metal used in batteries, pigments, metal coatings, and plastics, has been linked to reduced sperm motility and to gynecological disorders such as endometriosis.⁷³

Reproductive health, fertility and TSCA

In June 2009, The Endocrine Society, a professional association devoted to research on hormones and the clinical practice of endocrinology, issued a scientific statement on endocrine disrupting chemicals which found:⁷⁴

The evidence for adverse reproductive outcomes (infertility, cancers, malformations) from exposure to endocrine disrupting chemicals is strong, and there is mounting evidence for effects on other endocrine systems, including thyroid, neuroendocrine, obesity and metabolism, and insulin and glucose homeostasis.

Among the statement's recommendations for the future is this suggestion:

As endocrinologists, we suggest that The Endocrine Society actively engages in lobbying for regulation seeking to decrease human exposure to the many endocrine-disrupting agents.

In November 2009, the American Medical Association (AMA) House of Delegates passed a resolution introduced by The Endocrine Society that calls for the AMA to work with the federal government to enact new federal policies to decrease the public's exposure to endocrine-disrupting chemicals.



These “new federal policies” and “regulation to decrease human exposure” would come through effective reform of TSCA. No chemicals are currently regulated under TSCA because of their potential to harm reproduction or development. But other authoritative bodies have listed more than 50 industrial chemicals as reproductive toxins.⁷⁵ TSCA reform should prioritize action on these chemicals and others such as bisphenol A, phthalates, and perfluorinated chemicals, which have been identified as harmful to reproduction and development.⁷⁶

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THE COST TO OUR HEALTH

Asthma

Asthma is the most common childhood chronic disease. The number of people with asthma roughly doubled between 1980 and 1995 in the U.S.⁷⁷ In 2008, more than 38 million people in the U.S. had experienced asthma at some point during their lives. Of these, ten million are children.^{78,79} In 2008, almost one in 10 (9.4% or seven million) children in the U.S. had asthma. Non-Hispanic black children are more likely to have asthma (16%) than Hispanic children (7%) or non-Hispanic white children (9%).⁸⁰ The annual cost of asthma is estimated to be nearly \$18 billion, with \$10 billion of that total being direct medical costs and \$8 billion in lost earnings due to illness or death.⁸¹

The link to chemical exposure

The doubling of asthma rates over the last two decades has prompted researchers to examine the role that chemicals and other environmental risk factors may play in this trend. Genetics cannot explain such dramatic increases in prevalence over such a short time. Extensive evidence from occupational studies and general population epidemiological and medical case reports documents that hundreds of chemicals can cause asthma in individuals previously free of the disease or can place asthma patients at greater risk for subsequent attacks.^{82,83}

In his 2007 review, Mark J. Mendell surveyed the literature to find 21 studies that link indoor residential chemical emissions and respiratory health or allergy problems in infants or children. He identified formaldehyde or particle-board, phthalates or plastic materials, and recent painting as the most frequent risk factors. Elevated risks were also reported for renovation and cleaning activities, new furniture, and carpets or textile wallpaper. Table 3 provides an overview of the indoor sources identified in Mendell's paper.

TABLE 3 Indoor Sources for Chemical Pollutants

Compounds	Example Sources
Aldehydes	
Formaldehyde	Composite wood and other products with urea-formaldehyde resin, some architectural finishes, tobacco smoke, and other combustion processes (carpet, paint)
Aromatics	
Benzene, toluene, xylenes, styrene, ethylbenzene, ethyltoluenes, and naphthalene	Motor vehicle exhaust, gasoline/fuel, tobacco smoke, solvent-based paints, floor adhesives, PVC flooring, carpeting, printed material, solvent-based consumer products
Dichlorobenzene	Moth balls, bathroom deodorizers
Chlorobenzene	Possibly solvent-based paints
Aliphatic hydrocarbons	
Hexane, nonane, decane, undecane, and dodecane	Some architectural finishes, floor adhesives, PVC flooring, consumer products (waxes, aerosol air fresheners)
Aliphatics (general)	Carpet padding, adhesives, calks, consumer products (paint)
VOCS, other	
Methylcyclopentane	Motor vehicle exhaust and evaporative emissions (carpets)
Butanol	Some architectural finishes
Limonene	Cleaning products, air fresheners, many consumer products
Tetrachloroethylene	Dry-cleaning solvent and dry-cleaned clothing (renovation)
Trichloroethylene	Aerosol paints, adhesives, lubricating oils, paint removers
Phthalate esters	
BBZP	Vinyl flooring, carpet tile, adhesives
DEHP	Vinyl flooring, PVC plastics

The 21 studies in the Mendell review include a 2004 Swedish study that compared 198 young children with asthma and allergies to 202 healthy control subjects. The home environment of every child was examined, with air and dust samples taken in the room where the child slept. The children whose bedrooms contained higher levels of the phthalate

DEHP were more likely to have been diagnosed with asthma by a physician.⁸⁴

Asthma and TSCA

Consumers, retailers, and other downstream users of chemicals (including manufacturers of and distributors of toys and other products) have a problem in common: They cannot gain access to



basic information about the chemicals used to make their products. Because TSCA does not ensure the right to know, we don't have the information we need to identify all the sources of indoor air pollution that are causing and contributing to asthma.

How can an expectant mother determine if there is formaldehyde in the particle-board used to make cribs and other nursery furnishings? How does a new father decide which strollers may contain and release phthalates? Why should new parents even have to worry about whether dangerous chemicals are in the products they choose for their newborn children?

TSCA reform should require chemical manufacturers to disclose what they know about chemical uses, and for clearly hazardous substances, chemical manufacturers should also have to disclose the products in which they are used.⁸⁵

Consumers, retailers, and other downstream users of chemicals (including manufacturers of and distributors of toys and other products) have a problem in common: They cannot gain access to basic information about the chemicals used to make their products. Because TSCA does not ensure the right to know, we don't have the information we need to identify all the sources of indoor air pollution that are causing and contributing to asthma.

Our Health Care Costs

“If our students are getting sick because we’ve built schools in polluted areas, they are going to fall behind. The poor who get sick because of toxins in their neighborhoods are the same people who typically seek treatment in emergency rooms. That drives up health care costs for everyone. And environmental health issues hold back economic growth. Let me repeat that, because there are a lot of people who think that we can’t address these issues and strengthen our economy. In fact, we must address these issues to strengthen our economy. Environmental health issues hold back economic growth.”

— EPA Administrator Lisa Jackson at the American Public Health Association, November 8, 2009⁸⁶

Administrator Jackson offered up a common sense equation in her speech to the American Public Health Association: A decline in exposure to toxic chemicals will result in a decline in chronic disease and a lowering of health care costs.

While it is exceedingly difficult to quantify how much money would be saved by implementing a specific legislative or policy regimen, some sense of the potential for savings can be gleaned from economic analyses of related reform efforts.

In 2003, the European Commission, the Executive Branch of the European Union, calculated the health care savings that would be attained by modernizing European chemicals management. The European Commission estimated that its proposed chemical management reforms would reduce health care costs by one-tenth of one percent in Europe.⁸⁷ Applying the European Commission’s formula to the U.S. Department of Health and Human Services’ National Health Expenditure Projections⁸⁸ yields an estimate that U.S. chemicals policy reform could save at least \$5 billion in health care costs each year.

Other analyses suggest that the European Commission calculations significantly underestimate the potential health care savings. In 2000, an expert committee of the U.S. National Academy of Sciences’ National Research Council concluded that 3% of developmental disabilities

TABLE 4 TSCA Reform and Health Care Costs: Estimates of Costs and Savings

	Costs in \$ Billions
Cancer: Direct Medical Costs 2008	\$93.2 billion ⁸⁹
Alzheimer’s Annual Cost of Care	\$150 billion ⁹⁰
Parkinson’s Disease Midpoint Annual Cost	\$20.8 billion ⁹¹
Infertility Treatment 2002	\$2.9 billion ⁹²
Hysterectomy for Treatment of Fibroids Annually	\$1.7 billion ⁹³
Preterm Births 2005	\$26.2 billion ⁹⁴
Asthma Direct Medical Cost	\$10 billion ⁹⁵
Total of Listed Health Costs	\$304.8 billion
10% of Listed Health Costs	\$30.48 billion
Mt Sinai Annual Savings**	\$2.3 billion ⁹⁶
Environment Canada***	\$6.9 billion ⁹⁷
Projected National Health Expenditure 2020	\$5000 billion
.1% of Projected 2020 Expenditure	\$5 billion

** Childhood cancer, asthma and developmental disabilities only

*** Diabetes, Parkinson’s disease and neurodevelopmental disabilities only

are the direct result of exposure to industrial chemicals and another 25% arise from interactions between environmental factors and genetic susceptibility.⁹⁸

In 2001, a study performed for Environment Canada estimated that the U.S. spends more than \$6.9 billion each year

in health care costs just for the 10% of diabetes, Parkinson’s disease, and neurodevelopmental effects it attributed to environmental contaminants.⁹⁹

In 2002, researchers from New York’s Mt. Sinai School of Medicine Center for Children’s Health and the Environment estimated that 5% of cancer, 10%



Applying the European Commission's formula to the U.S. Department of Health and Human Services' National Health Expenditure Projections yields an estimate that U.S. chemicals policy reform could save at least \$5 billion in health care costs each year.

of neurobehavioral disorders and 30% of asthma in children are associated with toxic chemicals found in our air, food, water, and the places we live, work, study and play.¹⁰⁰ The 2002 Mt. Sinai report calculated that more than \$2.3 billion are spent every year just on the medical costs of childhood cancer, asthma and neurobehavioral disorders associated with toxic chemicals.

Table 4 shows the available data on the costs of cancer, Parkinson's and Alzheimer's disease, reproductive health problems, and asthma to provide a range of possible cost savings estimates. If TSCA reform led to toxic chemical exposure reductions that saved 10% of these listed costs, the U.S. could save more than \$30 billion annually in current dollars.

While the estimation method we have used for these calculations is entirely different from that of the European Commission, the range of estimates supports the argument that if TSCA reform leads to reductions in toxic chemical exposures that translate into just a tenth of one percent reduction in health care costs, the U.S. healthcare system will save at least \$5 billion every year.

Conclusion

When the Toxics Substances Control Act became law in 1976, smoking was permitted in airplanes, hospitals, and all other public places. There were no laws requiring that children or adults wear seat belts. Lead was still being added to gasoline. In the more than 30 years that have passed, Americans and their elected officials have taken action on all of these

issues and witnessed significant improvement in public health.

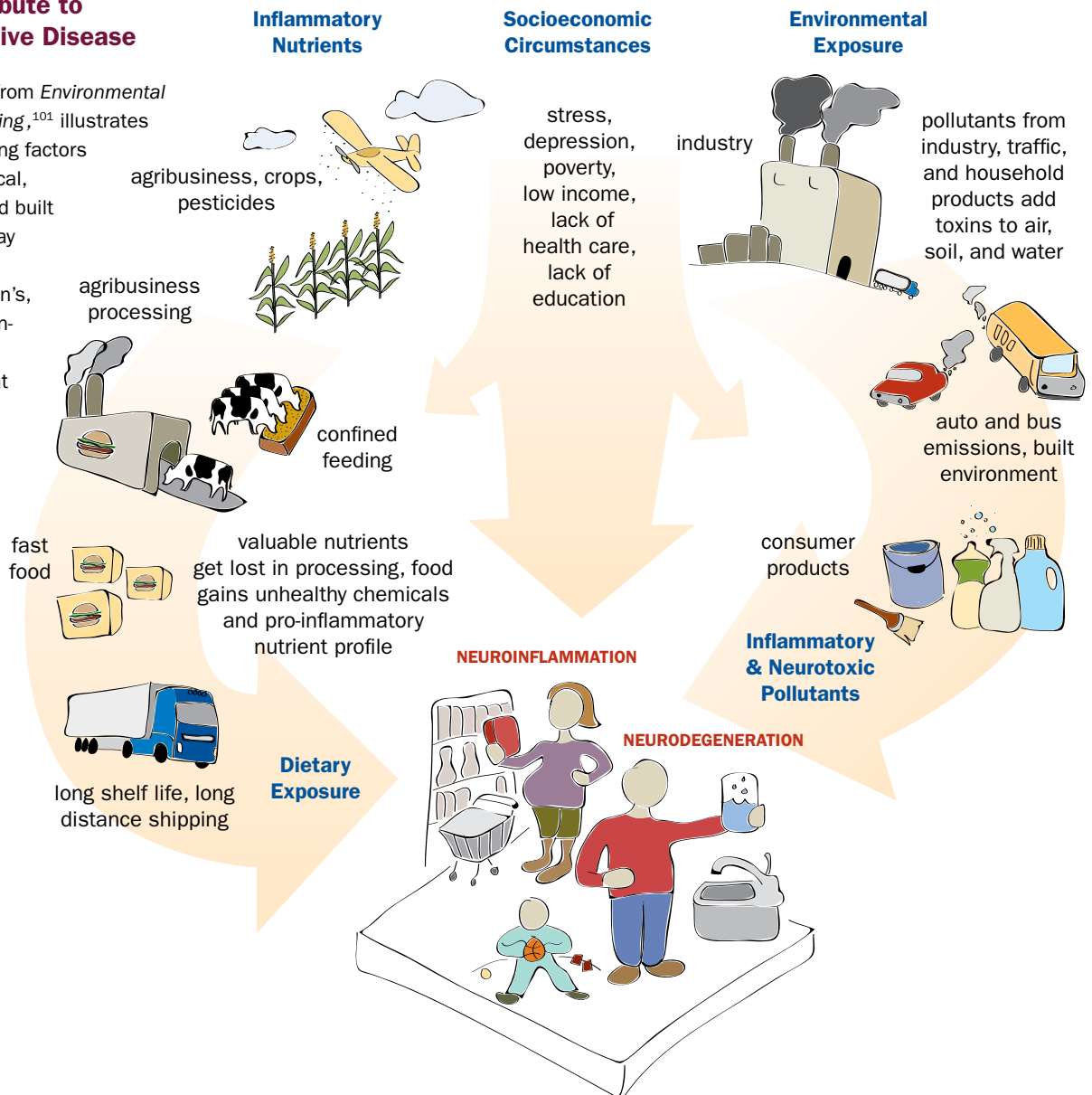
The last 30 years of science linking human health and the environment make clear that TSCA reform presents another opportunity to dramatically improve public health:

1. The new science on cancer shows why TSCA reform should require

chemical manufacturers to provide basic information on the health hazards associated with their chemicals, how they are used, and the ways that the public or workers could be exposed. Chemical manufacturers need to be made responsible for demonstrating the safety of their products. The law should be designed to protect the health of all Americans, by ensuring EPA has ample authority

FIGURE 3 Interacting Factors that May Contribute to Neurodegenerative Disease

This figure, adapted from *Environmental Threats to Healthy Aging*,¹⁰¹ illustrates some of the interacting factors in the modern chemical, nutritional, social, and built environments that may be contributing to Alzheimer's, Parkinson's, and other neurodegenerative disease. New science suggests that most other chronic disease is also the result of interacting factors.



to take action on dangerous chemicals like asbestos.

2. Recent research demonstrating the ability of relatively small amounts of chemicals to permanently harm the developing brain illustrates the critical need to protect especially vulnerable people. Effective TSCA reform must ensure that all existing and new chemicals are safe for pregnant women, children, workers and other sensitive populations.
3. The studies suggesting linkages between chemical exposures and Parkinson's and Alzheimer's disease highlight that effective TSCA reform must recognize the unique dangers posed by exposure to persistent, bioaccumulative toxins (PBTs), like PCBs, and include provisions to phase out all non-essential uses of them. Communities and populations that bear disproportionately high burdens of PBT contamination need to be the focus of exposure reduction efforts.
4. The findings on reproductive health and fertility problems make the case for prioritized action on bisphenol A, phthalates, perfluorinated compounds, and other industrial chemicals that new science identifies as harmful to reproduction and development.
5. And the growing body of research linking asthma to chemical exposures shows why the public, workers, and the marketplace should have full access to information about the health and environmental hazards of chemicals, how they are used, and the ways that exposure might take place.

Figure 3 from *Environmental Threats to Healthy Aging*¹⁰² illustrates the interacting factors that can lead to the symptoms of Alzheimer's and Parkinson's disease.

In Lisbeth B. Schorr's 1988 book *Within Our Reach—Breaking the Cycle of Disadvantage*,¹⁰³ she describes the stressors or risk factors for “rotten outcomes” for America's disadvantaged children



In simplest terms, real reform will lead to more healthy babies, fewer women with breast cancer, a return to normal fertility patterns, and lower numbers of people with Alzheimer's disease. This is the promise of TSCA reform.

and the elements of successful programs to prevent those rotten outcomes.

Schorr explains that “it takes multiple and interacting risk factors to produce damaging outcomes. Lasting damage occurs when the elements of a child's environment—at home, at school, in the neighborhood—multiply each other's destructive effect.”

But just as the adding on of risk factors can multiply harm, the removal of risk factors can divide the negative impact. Schorr explains:

The implication is clear: The prevention of rotten outcomes is not a matter of all or nothing. It will be

of value if we can eliminate one risk factor or two, even if others remain. By distinguishing between those factors we can do something about and those we can't, the problem becomes less intractable.

Over the last three decades scientists have learned that chronic disease is also the result of “multiple and interacting risk factors.” Exposure to chemicals is such a factor, along with poor nutrition, lack of exercise, genes that predispose one to disease, infection, challenging social and economic conditions, age, and tobacco use. The interplay of variables begins before conception and continues for an entire lifetime.

Chemical exposure is a factor we can do something about. By reforming TSCA, we can lessen the role of chemical exposures in causing disease, thereby reducing our nation's chronic disease burden and helping to control health care costs. In simplest terms, real reform will lead to more healthy babies, fewer women with breast cancer, a return to normal fertility patterns, and lower numbers of people with Alzheimer's disease. This is the promise of TSCA reform.

Endnotes

- 1 U. S. Environmental Protection Agency, "EPA Administrator Jackson Unveils New Administration Framework for Chemical Management Reform in the United States," <http://yosemite.epa.gov/opa/admpress.nsf/0/D07993FDC8F01C-2285257640005D27A6> (accessed November 8, 2009).
- 2 Tracey J. Woodruff, et al., *America's Children and the Environment*, (Washington, DC: U.S. Environmental Protection Agency, 2008).
- 3 Holly L. Howe, et al., "Annual Report to the Nation on the Status of Cancer (1973 through 1998), Featuring Cancers with Recent Increasing Trends," *Journal of the National Cancer Institute*, 93, no. 11 (June 2001): 824-42.
- 4 Janet Gray, ed, *State of the Evidence: The Connection Between Breast Cancer and the Environment*, (San Francisco: Breast Cancer Fund, 2008).
- 5 Tracey J. Woodruff, et al., "Trends in Environmentally Related Childhood Illnesses," *Pediatrics*, 113, no. 4 (April 2004): 1133-1140.
- 6 Jeanne E. Moorman, et al., "National Surveillance for Asthma, United States 1980-2004," Centers for Disease Control and Prevention, <http://www.cdc.gov/mmwr/preview/mmwrhtml/ss5608a1.htm> (November 1, 2009).
- 7 Anjani Chandra and Elizabeth Hervey Stephen, "Impaired Fecundity in the United States: 1982-1995," *Family Planning Perspectives*, 30, no 1, (1998): 34-42.
Anjani Chandra, et al., "Fertility, Family Planning and Reproductive Health of US Women: Data from the 2002 National Survey of Family Growth," *Vital and Health Statistics*, 23, no. 25 (2005).
- 8 Kate Brett, "Fecundity in 2002 National Survey of Family Growth Women 15-24 Years of Age", Hyattsville, MD, National Center for Health Statistics (2008).
- 9 Leonard J. Paulozzi, "International Trends in Rates of Hypospadias and Cryptorchidism," *Environmental Health Perspectives*, 107, no. 4, (1999): 297-302.
- 10 National Institute of Mental Health, "NIMH's Response to New Autism Prevalence Estimate," <http://www.nimh.nih.gov/about/director/updates/2009/nimhs-response-to-new-autism-prevalence-estimate.shtml> (November 4, 2009).
- 11 National Center for Chronic Disease Prevention and Health Promotion, *The Power of Prevention: Chronic Disease...the Public Health Challenge of the 21st Century*, (Washington, DC: Centers for Disease Control and Prevention, 2009).
- 12 Commission of the European Communities, "Regulation of the European Parliament and of the Council Concerning the Registration, Evaluation, Authorisation and Restrictions of Chemicals (REACH), establishing European Chemicals Agency and Amending Directive 1999/45/EC and Regulation (EC) on Persistent Organic Pollutants: Extended Impact Assessment." (October 29, 2003): 30.
- 13 Philip J. Landrigan, et al., "Environmental Pollutants and Disease in American Children: Estimates of Morbidity, Mortality, and Costs for Lead Poisoning, Asthma, Cancer, and Developmental Disabilities," *Environmental Health Perspectives*, 110, No. 7 (July 2002): 721-8.
- 14 Tom Muir and Mike Zegarac, "Societal Costs of Exposure to Toxic Substances: Economic and Health Costs of Four Case Studies That Are Candidates for Environmental Causation," *Environmental Health Perspectives Supplements*, 109, No. S6 (December 2001): 885-903.
- 15 Philip J. Landrigan, et al., "Environmental Pollutants and Disease in American Children: Estimates of Morbidity, Mortality, and Costs for Lead Poisoning, Asthma, Cancer, and Developmental Disabilities," *Environmental Health Perspectives*, 110, No. 7 (July 2002): 721-8.
- 16 National Center for Chronic Disease Prevention and Health Promotion, *The Power of Prevention: Chronic Disease...the Public Health Challenge of the 21st Century*, (Washington, DC: Centers for Disease Control and Prevention, 2009).
- 17 National Cancer Institute, Surveillance Epidemiology and End Results Program, "SEER Stat Fact Sheets," <http://seer.cancer.gov/statfacts/html/all.html> (accessed October 5, 2009).
- 18 National Cancer Institute, Surveillance and Epidemiology End Results Program, "Data for Invasive Cancers," http://seer.cancer.gov/csr/1975_2006/browse_csr.php?section=2&page=sect_02_table.11.html (accessed October 5, 2009).
- 19 American Cancer Society. *Cancer Facts & Figures 2009* (Atlanta: American Cancer Society, 2009).
- 20 American Cancer Society, "Costs of Cancer," http://www.cancer.org/docroot/MIT/content/MIT_3_2X_Costs_of_cancer.asp (accessed October 5, 2009).
- 21 Philip J. Landrigan, et al., "Environmental Pollutants and Disease in American Children: Estimates of Morbidity, Mortality, and Costs for Lead Poisoning, Asthma, Cancer, and Developmental Disabilities," *Environmental Health Perspectives*, 110, No. 7 (July 2002): 721-8.
- 22 *Report on Carcinogens, Eleventh Edition*; U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program.
- 23 Ibid.
- 24 Sydney M. Gordon, et al., "Residential Environmental Measurements in the National Human Exposure Assessment Survey (NHEXAS) Pilot Study in Arizona: Preliminary Results for Pesticides and VOCs," *Journal of Exposure Analysis and Environmental Epidemiology*, 9 (1999): 456-470.
- 25 Agency for Toxic Substances and Disease Registry, *Toxicological Profile for Formaldehyde*, (Washington, DC: Department of Health and Human Services, 1999).
- 26 US Environmental Protection Agency, *Trichloroethylene (TCE): TEACH Chemical Summary* (Washington, DC: US Environmental Protection Agency, 2007).
- 27 Agency for Toxic Substances and Disease Registry, *Toxicological Profile for Trichloroethylene (TCE)*, (Washington, DC: Department of Health and Human Services, 1997).
- 28 Edo D. Pellizzari, et al., "Purgeable Organic Compounds in Mother's Milk," *Bulletin of Environmental Contamination and Toxicology*, 28, No. 23 (March 1982): 322-8.
- 29 Barbara A. Cohn, et al., "DDT and Breast Cancer in Young Women: New Data on the Significance of Age at Exposure," *Environmental Health Perspectives*, 115, No. 10 (October 2007): 1406-14.
- 30 Julia Green Brody, et al., "Environmental Pollutants and Breast Cancer: Epidemiologic Studies," *Cancer*, 109, No. 12 (June 12, 2007): 2667-2711.
- 31 Barbara A. Cohn, et al., "DDT and Breast Cancer in Young Women: New Data on the Significance of Age at Exposure," *Environmental Health Perspectives*, 115, No. 10 (October 2007): 1406-14.
- 32 Janet Gray, ed, *State of the Evidence: The Connection Between Breast Cancer and the Environment*, (San Francisco: Breast Cancer Fund, 2008).
- 33 SH Dairkee, et al., "Bisphenol A induces a profile of tumor aggressiveness in high-risk cells from breast cancer patients." *Cancer Research*, 68, no. 7(April 1, 2008): 2076-80.
- 34 International Ban Asbestos Secretariat, "Current Asbestos Ban Restrictions," http://ibasecretariat.org/lka_alpha_asb_ban_280704.php (accessed October 1, 2009).
- 35 Lowell Center for Sustainable Production, *The Promise and Limits of the United States Toxic Substances Control Act*, (Lowell, MA: Lowell Center for Sustainable Production, 2003).

- 36 This entire section is informed by the article "Environmental and Occupational Causes of Cancer: New Evidence 2005–2007." by R.W. Clapp, M.M. Jacobs, and E.L. Loechler, published in the journal *Reviews on Environmental Health*, 23, no. 1 (2008): 1–47.
- 37 Steven G. Gilbert, "The Scientific Consensus Statement on Environmental Agents Affiliated with Neurodevelopmental Disorders," (Bollinas, CA: Collaborative on Health and the Environment, 2008), abstracted in *Neurotoxicology and Teratology*, 31, no. 4, (July–August 2009): 241–2.
- 38 National Institute of Mental Health, "NIMH's Response to New Autism Prevalence Estimate," <http://www.nimh.nih.gov/about/director/updates/2009/nimhs-response-to-new-autism-prevalence-estimate.shtml> (November 4, 2009).
- 39 Catherine Rice, "Prevalence of autism spectrum disorders — Autism and Developmental Disabilities Monitoring Network, United States, 2006, National Center on Birth Defects and Developmental Disabilities," *MMWR Surveillance Summaries*, 58, no. SS10 (December 18, 2009): 1–20. <http://www.cdc.gov/mmwr/preview/mmwrhtml/ss5810a1.htm>.
- 40 Irva Hertz-Picciotto and Lora Delwiche, "The Rise in Autism and the Role of Age in Diagnosis," *Epidemiology*, 20, no. 1 (2009): 84–90.
- 41 Steven G. Gilbert, "The Scientific Consensus Statement on Environmental Agents Affiliated with Neurodevelopmental Disorders," (Bollinas, California: Collaborative on Health and the Environment, 2008), abstracted in *Neurotoxicology and Teratology*, 31, no. 4 (July–August 2009): 241–2.
- 42 Centers for Disease Control and Prevention, "CDC Statement on Autism Data," <http://www.cdc.gov/ncbddd/autism/data.html> (accessed October 10, 2009).
- Tom T. Shimabukuro, Scott D. Grosse SD, and Catherine Rice, "Medical Expenditures for Children with an Autism Spectrum Disorder in a Privately Insured Population," *Journal of Autism and Developmental Disorders*, 38, no. 3 (March 2008): 546–52.
- 43 M.L. Ganz, "The Costs of Autism," *Understanding Autism: From Basic Neuroscience to Treatment*, First Edition, Steven O. Molden, ed, and John Rubenstein, ed, (Boca Raton, Florida: CRC Press, 2006): 475–502.
- 44 This entire section is informed by the article "Developmental Neurotoxicity of Industrial Chemicals," by P. Grandjean and P.J. Landrigan, published in the journal *Lancet*, 368, no. 9553 (December 16, 2006): 2167–78.
- 45 Philippe Grandjean and Philip J. Landrigan, "Developmental Neurotoxicity of Industrial Chemicals," *Lancet*, 368, no. 9553 (December 2006): 2167–78.
- 46 Donald E. Schmechel, Jeffrey Brownkyke, Andrew Ghi, "Strategies for Dissecting Genetic-Environmental Interactions in Neurodegenerative Disorders," *Neurotoxicology*, 27, no. 5 (September 2006): 637–57.
- 47 Marc G. Weisskopf, et al., "Cumulative Lead Exposure and Prospective Change in Cognition among Elderly Men," *American Journal of Epidemiology*, 160, no. 12 (2004): 1184–93.
- 48 53 Jinfang Wu, et al., "Alzheimer's Disease (AD)-Like Pathology in Aged Monkeys after Infantile Exposure to Environmental Metal Lead (Pb): Evidence for a Developmental Origin and Environmental Link for AD," *The Journal of Neuroscience*, 28, no. 1 (2008): 3–9.
- 49 M. Riyaz Basha, Wei Wei, Saleh A. Bakheet, et al., "The Fetal Basis of Amyloidogenesis: Exposure to Lead and Latent Overexpression of Amyloid Precursor Protein and Beta-Amyloid in the Aging Brain," *The Journal of Neuroscience*, 25, no. 4 (2005): 823–829.
- 50 J.R. Walton, "A Longitudinal Study of Rats Chronically Exposed to Aluminum at Human Dietary Levels," *Neuroscience Letters*, 412 (2007): 29–33.
- 51 J.R. Walton, "Human Range Dietary Aluminum Equivalents Cause Cognitive Deterioration in Aged Rats," (presented at the 24th International Neurotoxicology Conference, San Antonio, Texas, November, 2007).
- 52 Kyle Steenland, Misty J. Hein, Rick Cassinelli, et al., "Polychlorinated Biphenyls and Neurodegenerative Disease Mortality in an Occupational Cohort," *Epidemiology*, 17, no. 1 (2006): 8–13.
- 53 Don M. Gash, et al., "Trichloroethylene: Parkinsonism and Complex 1 Mitochondrial Neurotoxicity," *Annals of Neurology*, 63, no. 2 (2007): 184–92.
- 54 Centers for Disease Control and Prevention, *Third National Report on Human Exposure to Environmental Chemicals*, Department of Health and Human Services, 2005.
- 55 This entire section is informed by *Environmental Threats to Healthy Aging: With a Closer Look At Alzheimer's and Parkinson's Disease*, by J. Stein, et al., (Boston: Greater Boston Physicians for Social Responsibility and the Science and Environmental Health Network, 2008).
- 56 Anjani Chandra and Elizabeth Hervey Stephen, "Impaired Fecundity in the United States: 1982-1995," *Family Planning Perspectives*, 30, no. 1 (1998): 34–42.
- Anjani Chandra, et al., "Fertility, Family Planning and Reproductive Health of US Women: Data from the 2002 National Survey of Family Growth," National Center for Health Statistics, *Vital Health Statistics*, 23, no. 25 (2005).
- 57 Thomas G. Travison, et al., "A Population-Level Decline in Serum Testosterone Levels in American Men," *The Journal of Clinical Endocrinology & Metabolism*, 92, no. 1 (2007).
- 58 Mona Shah, et al., "Trends in Testicular Germ Cell Tumours by Ethnic Group in the United States," *International Journal of Andrology*, 30 (2007): 206–13.
- 59 Shanna Swan, Eric P. Elkin, and Laura Fenster, "The Question of Declining Sperm Density Revisited: An Analysis of 101 Studies Published 1934–1996," *Environmental Health Perspectives*, 108, no. 10 (October 2000): 961–66.
- 60 Leonard J. Paulozzi, J. David Erickson, and Richard J. Jackson, "Hypospadias Trends in Two US Surveillance Systems," *Pediatrics*, 100, no. 5 (November 1997): 831–34.
- 61 M. Davidoff, et al., "Changes in the Gestational Age Distribution among U.S. Singleton Births: Impact on Rates of Late Preterm Birth, 1992 to 2002," *Seminars in Perinatology*, 30, no. 1 (2006): 8–15.
- 62 Susan W. Euling, et al., "Role of Environmental Factors in the Timing of Puberty," *Pediatrics*, 121, S3 (February 2008): S167–71.
- 63 The Collaborative on Health and the Environment, *Vallombrosa Consensus Statement on Environmental Contaminants and Human Fertility Compromise October 2005*, http://www.healthandenvironment.org/infertility/vallombrosa_documents (accessed September 28, 2009).
- 64 Richard E. Behrman and Adrienne Stith Butler, ed, "Committee on Understanding Premature Birth and Assuring Healthy Outcomes," *Preterm Birth: Causes, Consequences and Prevention*, (Washington, DC, Institute of Medicine of National Academies, 2006).
- 65 Jo M. Braun, et al., "Prenatal Bisphenol A Exposure and Early Childhood Behavior," *Environmental Health Perspectives*—in press (2009).
- 66 D. Li, et al., Occupational Exposure to Bisphenol-A (BPA) and the Risk of Self-Reported Male Sexual Dysfunction, *Human Reproduction*, published on-line 2009 Nov 10. [Epub ahead of print]
- 67 E. Diamanti-Kandarakis, et al. "Endocrine-Disrupting Chemicals: An Endocrine Society Scientific Statement," *Endocrine Reviews*, 30, no. 4 (2009): 293–342.
- 68 SH Swan et al., "Prenatal phthalate exposure and reduced masculine play in boys." *International Journal of Andrology*, (November 16, 2009) [Epub ahead of print]
- 69 Ivelisse Colón, et al., "Identification of Phthalate Esters in the Serum of Young Puerto Rican Girls with Premature Breast Development." *Environmental Health Perspectives*, 108, no. 9 (2000): 895–900.

- 70 L. Qiao, L. Zheng, D. Cai, [Study on the Di-N-Butyl Phthalate and Di-2-Ethylhexyl Phthalate Level of Girl Serum Related with Precocious Puberty in Shanghai], *Wei Sheng Yan Jiu*, 36 (2007): 93–95.
- 71 Benjamin J. Apelberg, Frank R. Witter, Julie B. Herbstman, et al., “Cord Serum Concentrations of Perfluorooctane Sulfonate (PFOS) and Perfluorooctanoate (PFOA) in Relation to Weight and Size at Birth,” *Environmental Health Perspectives*, 115, no. 11 (November 2007): 1670–6.
- 72 Ulla Nordström Joensen, et al., “Do Perfluoroalkyl Compounds Impair Human Semen Quality?” *Environmental Health Perspectives*, 177, no. 6 (June 2009).
- 73 Tracey J. Woodruff, et al., “Proceedings of the Summit on Environmental Challenges to Reproductive Health and Fertility: Executive Summary,” *Fertil Steril*, 89, no. 2 (February 2008): 281–300.
- 74 Ibid.
- 75 California Environmental Protection Agency, “Proposition 65: The Safe Drinking Water and Toxic Enforcement Act of 1986,” Chemicals Known to the State to Cause Cancer or Reproductive Toxicity List as of September 11, 2009.
- 76 This entire section is informed by the article “Female Reproductive Disorders: the Roles of Endocrine-Disrupting Compounds and Developmental Timing,” by D. Andrew Crain, et al., *Fertility and Sterility*, 90, no. 4 (2008): 911–40.
- 77 Tracey J. Woodruff, Daniel A. Axelrad DA, Amy D. Kyle, Onyemaechi Nweke, Gregory G. Miller, and Bradford J. Hurley, “Trends in Environmentally Related Childhood Illnesses,” *Pediatrics*, 113, no. 4 (April 2004): 1133–1140.
- 78 National Health Interview Survey, “Summary Health Statistics for U.S. Adults” *Vital and Health Statistics*, Series 10, no. 242 (2008).
- 79 National Health Interview Survey, “Summary Health Statistics for U.S. Children” *Vital and Health Statistics*, Series 10, no. 244 (2008).
- 80 Ibid.
- 81 Asthma and Allergy Foundation of America, “Asthma Facts and Figures,” <http://www.aafa.org/display.cfm?id=8&sub=42> (accessed August 12, 2009).
- 82 Jean-Luc Malo and Mora Chan-Yeung, “Appendix: Agents Causing Occupational Asthma with Key References,” in *Asthma in the Workplace, Third Edition*, eds, I Leonard Bernstein, Mora Chan-Yeung M, Jean-Luc Malo, and David I. Bernstein, (New York: Taylor & Francis, 2006).
- 83 Association of Occupational and Environmental Clinics, “AOEC Exposure Codes,” <http://www.aoec.org/aoeccode.htm> (accessed August 12, 2009).
- 84 Carl-Gustaf Bornehag, et al., “The Association between Asthma and Allergic Symptoms in Children and Phthalates in House Dust: A Nested Case–Control Study,” *Environmental Health Perspectives*, 112, no. 14 (October 2004): 1393–1397.
- 85 This entire section is informed by the article “Indoor Residential Chemical Emissions as Risk Factors for Respiratory and Allergic Effects in Children: A Review,” by Mark J. Mendell, *Indoor Air*, 17, no. 4 (2007): 259–77.
- 86 Lisa Jackson, “Remarks to the American Public Health Association, as Prepared for Delivery,” U.S. Environmental Protection Agency, <http://yosemite.epa.gov/opa/admpress.nsf/12a744ff56dbff8585257590004750b6/2af3d0143020edc1852576690052a953?OpenDocument> (accessed November 16, 2009).
- 87 Commission of the European Communities, “Regulation of the European Parliament and of the Council Concerning the Registration, Evaluation, Authorisation and Restrictions of Chemicals (REACH), Establishing European Chemicals Agency and Amending Directive 1999/45/EC and Regulation (EC) on Persistent Organic Pollutants: Extended Impact Assessment.” (October 29, 2003): 30.
- 88 Center for Medicaid and Medicare Services, “National Health Expenditure Data: Projected,” http://www.cms.hhs.gov/NationalHealthExpendData/03_NationalHealthAccountsProjected.asp#TopOfPage (accessed September 28, 2009).
- 89 American Cancer Society. *Cancer Facts & Figures 2009* (Atlanta: American Cancer Society, 2009).
- 90 Jill Stein, et al., *Environmental Threats to Healthy Aging: With a Closer Look At Alzheimer’s and Parkinson’s Disease*, ed. Nancy Myers (Boston: Greater Boston Physicians for Social Responsibility and the Science and Environmental Health Network, 2008).
- 91 Ibid.
- 92 The Collaborative on Health and the Environment, *Vallombrosa Consensus Statement on Environmental Contaminants and Human Fertility Compromise October 2005*, http://www.healthandenvironment.org/infertility/vallombrosa_documents (accessed September 28, 2009).
- 93 D. Andrew Crain, et al., “Female Reproductive Disorders: the Roles of Endocrine-Disrupting Compounds and Developmental Timing,” *Fertility and Sterility*, 90, no. 4 (2008): 911–40.
- 94 Richard E. Behrman and Adrienne Stith Butler, ed, “Committee on Understanding Premature Birth and Assuring Healthy Outcomes,” *Preterm Birth: Causes, Consequences and Prevention*, (Washington, DC, Institute of Medicine of National Academies, 2006).
- 95 Asthma and Allergy Foundation of America, “Asthma Facts and Figures,” <http://www.aafa.org/display.cfm?id=8&sub=42> (accessed August 12, 2009).
- 96 Philip J. Landrigan, et al., “Environmental Pollutants and Disease in American Children: Estimates of Morbidity, Mortality, and Costs for Lead Poisoning, Asthma, Cancer, and Developmental Disabilities,” *Environmental Health Perspectives*, 110, No. 7 (July 2002): 721–8.
- 97 American Cancer Society, “Costs of Cancer,” http://www.cancer.org/docroot/MIT/content/MIT_3_2X_Costs_of_cancer.asp (accessed October 5, 2009).
- 98 National Research Council, *Scientific Frontiers in Developmental Toxicology and Risk Assessment* (Washington DC: National Academy Press, 2000).
- 99 Tom Muir and Mike Zegarac, “Societal Costs of Exposure to Toxic Substances: Economic and Health Costs of Four Case Studies That Are Candidates for Environmental Causation,” *Environmental Health Perspectives Supplements*, 109, no. S6 (December 2001): 885–903.
- 100 Philip J. Landrigan, et al., “Environmental Pollutants and Disease in American Children: Estimates of Morbidity, Mortality, and Costs for Lead Poisoning, Asthma, Cancer, and Developmental Disabilities,” *Environmental Health Perspectives*, 110, no. 7 (July 2002): 721–8.
- 101 Jill Stein, et al., *Environmental Threats to Healthy Aging: With a Closer Look at Alzheimer’s and Parkinson’s Disease*, ed. Nancy Myers (Boston: Greater Boston Physicians for Social Responsibility and the Science and Environmental Health Network, 2008).
- 102 Ibid.
- 103 Lisbeth Schorr, *Within Our Reach: Breaking the Cycle of Disadvantage* (New York: Anchor Press, 1988).



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